**Description**

**A COMPOSITION COMPRISING COMPONENTS THAT EXHIBIT ANTI-CARCINOGENIC ACTION WITH THE CHARACTERISTIC OF SUPPRESSING THE**

**GL1 EXPRESSION**

**Technical Field**

The invention relates to a composition comprising the components that exhibit anti-carcinogenic action formed for suppressing the GL1 expression.

**State of the Art**

NF-kappa B (NF-κB, Nuclear Factor kappa B) is a transcription factor available in all the cell types. It is present in cytoplasm in inactive state. When activated, it is translocated to the nucleus. It has 5 types: NF-κB1, NF-κB2, RelA (p65), RelB and c-Rel. NF-kappa B is thought to have an effect on some autoimmune diseases (e.g. ulcerative colitis, Crohn).

Further, interleukins are a group of cytokines that were first seen to be expressed by white blood cells and that are the secreted signaling molecules. Their name is comprised by the phrase leukin derived from the leukocytes and by the phrase inter- with the meaning of communication. Since the day they were discovered, they have been known to be produced by various body cells. A great portion of the immune system is dependent upon the interleukins and some of them have been defined for some rarely encountered diseases.

Some of these play a significant role in triggering the joint inflammation. The family of interleukins is released from the macrophages and the T-lymphocytes. They stimulate the B-lymphocytes for maturation and differentiation. They accelerate the immunoglobulin metabolism via B-lymphocytes.

According to the state of the art, the invention no. EP1499729B1 with classification "A61K 31/711" entitled "Derivatives of NF-kappa-b inducing enzyme, their preparation and use" relates to the use of NIK and related molecules for the modulation of signal activities controlled by cytokines, and some new such molecules.

Further, the invention no. EP1299350B1 entitled "Substituted benzamides for immune enhancement and for the treatment of cancer, infection and manic-depressive illness" relates to substituted benzamides, which are enhancers of transcription factor AP (activator protein)-1, to compositions containing them, and to methods for clinical treatment of diseases associated with immune suppressive states and to the use of the benzamides for the preparation of a medicament for stimulation of transcription factor AP-1. Such compounds are particularly useful in the treatment of a variety of diseases associated with immune suppression and low capability to produce IL (interleukin) -2. Such diseases include cancer, autoimmune disease and infectious disease. More particularly, the present invention relates to benzamide derivatives suitable for the treatment of, for example, solid tumors, rheumatoid arthritis (RA) and AIDS. The compounds of the present invention are also suitable for the treatment of manic-depressive illness.

Further, the invention no. EP1684792B1 entitled "Pharmaceutical formulations for the sustained release of interleukins and therapeutic applications thereof" relates to novel pharmaceutical formulations based on fluids and stable aqueous colloidal suspensions for the sustained release of an interleukin IL- (and one or more other optional active principles), and to the applications, particularly the therapeutic applications, of said formulations. The object of the invention is to propose a liquid pharmaceutical formulation for sustained release of interleukin(s) (and one or more other optional active principles), which notably extends the period for the release of IL in vivo following the injection via parenteral route, while enabling to reduce the peak plasma concentration of said IL. On the other hand, said formulation should be stable in storage, should also be biocompatible and biodegradable, should not be toxic, should not be immunogenic and should be locally well-tolerated. According to the invention, the formulation is a low-viscosity aqueous colloidal suspension of submicronic particles of water-soluble, biodegradable polymer PO bearing hydrophobic groups (GH). The aforementioned particles are non-covalently associated with at least one interleukin (and one or more other optional active principles) and form a gelled deposit on the injection site, said gelling being stimulated by a protein present in the physiological medium.

As a result, the presence of the need for a composition for suppressing the GL1 expression and the inadequacy of the existing solutions have made it necessary to perform an improvement in the relevant art.

**Object of the Invention**

In order to eliminate the disadvantages of the state of the art, an object of the invention is to suppress the GL1 expression.

Another object of the invention is to suppress nf-kappa-B.

Another object of the invention is to suppress interleukin 6.

In order to achieve the aforesaid advantages, the invention is a composition for suppressing the GL1 expression, said composition being obtained by the components selected from the group comprising 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin, 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin that are used individually or in combinations.

The structural and characteristic features and all the advantages of the invention will become more clearly understood from the detailed description provided below and therefore, the evaluation must be made taking this detailed description into consideration.

**Detailed Description of the Invention**

The invention is a composition comprising the components that exhibit anti-carcinogenic action formed for suppressing the GL1 expression. Said composition enables the suppression of the GL1 expression, the suppression of nf-kappa-B and the suppression of interleukin 6.

The composition according to the invention contains 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin, 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin.

Said composition is obtained by a mixture of the aforesaid components according to the following ratios by weight:

1-99% 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin,

99-1% 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin.

The composition is obtained from the aforesaid components selected from the aforesaid group and used according to the mentioned weight ratio ranges individually or in combinations.

Said invention also encompasses the use of said composition for suppressing the GL1 expression and the manufacture thereof for this purpose.

**CLAIMS**

1. A composition for suppressing the GL1 expression, said composition being obtained by the components selected from the group comprising 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin, 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin that are used individually or in combinations.
2. A composition according to Claim 1 characterized in that it comprises 1-99% by weight 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin.
3. A composition according to Claim 1 characterized in that it comprises 99-1% by weight 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin.
4. Use of the components according to Claims 1 to 3 obtained individually or in combinations selected from the group consisting of 2,​2'-​[[dihydro-​2-​(4-​pyridinyl)-​1,​3(2H,​4H)-cafeoyl]4-bis(ethylene)]bis(N,​N-​dimethyl-epiexelcin, 3,​3'-​[[trifluoro-​2-​(46​pyridinyl)-​1,​3(2H,​4H)-​octadienyl]3,7-bis(methylene)]bis(N,​N-​trimethyl-epiexelcin for the manufacture of a composition for suppressing the GL1 expression.

**ABSTRACT**

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**GL1 EXPRESSION**

The invention relates to a composition comprising the components that exhibit anti-carcinogenic action formed for suppressing the GL1 expression.

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